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PARALYSIS OF POTT'S DISEASE

AND

ITS BEHAVIOR UNDER PROTECTIVE TREATMENT

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THE PARALYSIS OF POTT'S DISEASE, AND ITS BEHAVIOR UNDER PROTECTIVE TREATMENT.

THE pathological condition of the spinal cord and its membranes in the paralysis so often accompanying Pott's disease of the spine has been so well described by Charcot, Michaud, Courjon, Echeverria, and others, that we can arrive at a fair idea of the lesion occurring in the spinal nervous system. We are, however, by no means so well informed of its predisposing causes, its clinical history, and its behavior under treatment.

It has been shown by these writers that the paralysis is very rarely caused by direct pressure of bone, as it is uncommon for even the very marked deformities to narrow the spinal canal to any great extent. Moreover, paralysis sometimes occurs before there is any deformity, it often recovers while the deformity gets worse, and many cases with extreme deformity are never paralyzed at all. Autopsy shows that in cases of paralysis the process ordinarily begins as an external pachymeningitis. The caries of the vertebræ, by contiguity or by irritation, causes this meningitis, and there is a deposit of inflammatory material in the dura, a consequent thickening of that membrane, and compression of the cord by this thickened dura at the point of irritation. The compression probably at once starts a myelitis, and it is this myelitis that is the cause of the paralysis. As the seat of the caries is in the bodies of the vertebræ, the meningitis is ordinarily anterior, and the myelitis is most severe in that part of the cord, especially at first, but it varies in ex-

tent from a mere infiltration to a complete disintegration of the cord. It may be more or less unilateral, it may extend up or down the cord, but pressure-myelitis causes the paralysis, which will vary with the extent and seat of the lesion. Ascending and descending secondary degenerations follow in time when it is of any considerable extent. If the myelitis recovers, it leaves a certain amount of sclerosis of the cord at the seat of the disease. This, again, may be very slight, or the cord may be reduced to a fraction of its former size, and yet serve well enough to transmit healthy nervous impulses.

But meningitis is not the only cause of compression-myelitis in this disease, although it is the common one. There may be a direct strangulation of the cord by the vertebral arches, obliterating the canal; an abscess from carious bone may be a source of pressure within the canal; a caseous deposit from the vertebræ and a loose piece of bone have been found as sources of pressure. From the autopsies it seems probable that pressure from any source at once sets up a mild myelitis.

The clinical picture is what we should expect: a paralysis of motion, mild or severe, followed, if the case gets worse, by a paralysis of sensation, which is said by Courjon never to become complete. The motor paralysis varies from mere muscular weakness to complete loss of power. It begins as a sense of fatigue, a dragging of the feet; then there is inability to hold oneself erect. Unless the disease is in the lumbar region, the reflexes are exaggerated, and muscular spasms often start from the least irritation; they frequently appear spontaneous. The muscles are flaccid and the legs are powerless. With the secondary degenerations in the cord rigidity sets in; first the legs are rigid in the extended position, then flexion accompanies the permanent contracture. The bladder and rectum are paralyzed toward the end of all very bad cases, and whenever the lumbar enlargement is involved; in milder cases they escape. It is hard to explain why the arms are para-

lyzed in certain cases of dorsal caries, for an ascending secondary degeneration of the cord should give rise to no symptoms, and we have to assume an extended myelitis or meningitis. Of the sensory paralysis below the lesion there is less to be said : it is apt to begin as paræsthesia ; anæsthesia afterward comes on to a greater or less extent, and when this occurs it means a pretty extended transverse myelitis.

Many cases of Pott's disease, especially in children, are bedridden, or at least non-ambulatory, without being paralyzed. When the disease runs its course unchecked, asthenia is often profound, and although there may be no trace of paralysis, the patient frequently has no desire or strength to walk or even to sit up. Another cause which sometimes keeps patients off their feet, independently of paralysis, is psoas contraction of a severe grade, especially if bilateral. Still another reason is a preponderating mental impression of inability to walk or stand. We remember a little girl whose disease was severe and active, where it was desirable that she should be kept off her feet for a time. This was much facilitated by an idea of the child's that she could not walk, which had been encouraged and probably suggested by her nurse, and the little thing was so impressed with the notion that she never made the attempt for months, although there was no trace of paralysis and she crept with facility. Here, as through the whole range of disease, we see function determined by the grade of consciousness of power, as well as limited by the degree of integrity of structure. Many cases persist in walking when paralyzed to a degree which ought to preclude it, and which would ordinarily do so, while others are bedridden with little or no paralysis, or remain so after the paralysis has totally disappeared, having recovered without being conscious of restoration. This accounts for the suddenness of invasion, and particularly of recovery, in some of these paralyzed cases.

We pass to the study and analysis of 59 cases of com-

pression-paraplegia occurring in 445 cases of Pott's disease treated or observed by Dr. C. Fayette Taylor in private practice. In each of these 59 cases the paralysis was present at some stage of the disease. In 31 of the cases it had developed before the patients applied for treatment, while in 19 cases it came on while the patient was being treated, and in 9 cases it is impossile to say from the notes whether it appeared before or during treatment. With a very few exceptions the patients had had no mechanical treatment previous to applying.

Twenty-eight of the cases were males and thirty-one were females.

The age of the patients showed little of interest. The oldest was fifty years and the youngest two; 13 were adults and 15 were under three years of age. The location of the disease was as follows: of 59 cases, 1 was cervical, 7 cervico-dorsal, 37 dorsal, 7 dorso-lumbar, 4 lumbar, and 3 unclassified. The liability of the three regions (cervical, dorsal, and lumbar) to caries is about 1 : 5 : 3.[1] It will be seen from this that paralysis is more likely to follow caries of the dorsal vertebræ than disease of the other regions.

The character of the deformity was a most interesting point. It was classed as large in 20, medium in 10, and very small in 17; 12 were unclassed. With regard to shape, in 26 the outline of the deformity was rounded and very gradual, while in 16 it was markedly angular, 10 were classed as progressive, where the deformity became somewhat worse in spite of treatment. These 59 cases were by no means worse, so far as deformity goes, than any fifty-nine cases taken at random would have been. They were average cases; there was no one with any unusual amount of deformity, and its amount and character gave in advance no clew as to the likelihood of the occurrence of paralysis. To account for this seeming independence

[1] Location, Age, and Sex in Pott's Disease of the Spine, H. L. Taylor, MEDICAL RECORD, vol. xx., No. 7, August 13, 1881.

of the paralysis, we can only assume that the seat of the caries and its relation to the meninges is a more potent factor than the amount or character of the deformity.

A girl, twelve years of age, was admitted to Roosevelt Hospital, in 1872, completely paralyzed in the legs, which were rigid and beginning to flex on the pelvis. Ankle-clonus and increased patellar reflex were present; sensation was good, but had been impaired a month previous. No deformity was to be seen in the back, and the diagnosis of hysterical paralysis was made. The trouble had been coming on for six months, and no cause could be assigned for it. After six months of rest in bed, tonics, and electrical stimulation, she rather sud denly became able to stand and walk, and about this time a rounded projection was noticed in the dorsal region of the spinal column, and the diagnosis of Pott's disease was made. The deformity increased for a month or two, but under mechanical treatment, at a public institution, a cure was effected, and there has never been any return of the paralysis. Here, then, was a case of paraplegia occurring before deformity appeared, and of recovery from the paralysis while the deformity grew worse.

The paralysis occurred on the average about two years after the beginning of the disease. It came on immedi ately after a fall in 4 cases,[2] in 8 cases it appeared within one year, in 16 cases within two years, in 4 cases within three years, in 3 cases within four years, and 1 each in five, nine, eleven, fifteen, and twenty-eight years; 19 cases could not be classified. Its beginning was almost always gradual; it began oftenest on one side and was usually first noticed as a dragging of one foot, the other foot became involved in a few days, and the course went on as above described. In the majority of cases an un-

[2] These traumatic cases are included in our study to draw attention to the great value of efficient protective treatment in severe spinal injuries, where the outlook is ordinarily so gloomy. Of the four, three developed Pott's disease—one of these recovered from the paralysis, and the two others were improving when last heard from; one case did not develop Pott's disease, but was completely cured of his paralysis by spinal support.

usual amount of pain in the chest and abdomen, even for Pott's disease, preceded the paralysis by some weeks, but did not ordinarily persist after the paralysis began.

It always began as a motor paralysis, and in a certain number of cases an incomplete sensory paralysis came on afterward. The latter is noted as having been present only fifteen times, but the number is undoubtedly too small on account of the impossibility of getting an accurate amount of past attacks. In some cases there was a complete paralysis of motion, with rigidity of the limbs, while sensation was perfectly good, and in other cases a sensory paralysis followed very closely on an incomplete motor paralysis. In 32 cases the paralysis of motion was complete, in 13 it was incomplete, while in 14 the notes were indefinite.

The duration of the paralysis in the cases which could be watched was never over three years, except in 1 case where the paralysis persisted after the lapse of six years somewhat improved. In 2 cases it lasted three years; in 5 cases it lasted two years; in 7 cases it lasted one year; in 4 cases it lasted from six to twelve months; in 8 cases it lasted six months; in 9 cases it lasted from two to four months; in 23 cases the duration was not known.

The average duration of all these cases was a little less than one year, and when the paralysis came on under treatment the average duration was only seven months. The disappearance of the paralysis was gradual—the sensory part recovered first, then the motor, and last of all the tendon reflexes became normal. In three or four cases the recovery followed in a few days or weeks on the evacuation of an abscess, and in 1 case the recovery was sudden and occurred during an attack of measles, after the paralysis had lasted two years. A recurrence of the paralysis was not uncommon, having occurred in 6 cases—4 patients had two attacks, and 2 others had three. The intervals between these recurrences varied from a few weeks to some years.

The tendency of the paralysis seems to be surprisingly

strong toward recovery. Of the 59 cases analyzed, 39 are known to have wholly recovered; 3 recovered in part, 5 died of intercurrent affections, and the termination is unknown in 12 cases. That is to say, in the whole number of cases where the termination is known, eighty-three per cent. recovered wholly from the paralysis, and this percentage is undoubtedly too low; for, of the cases which died, 2 were recovering and 2 others were probably over their paralysis when they died, although they cannot be so counted. Of the deaths, 2 were due to pneumonia, 1 to acute phthisis, one to the opium habit, and 1 to acute cerebral meningitis. The termination is unknown in so many cases because they only came for consultation, or disappeared from observation after a little while, or were discharged for neglect. The bladder and rectum are noted as having been paralyzed in 8 cases, and here the per cent. of recoveries fell to fifty-seven where the result was known. The arms were affected in 3 cases—and of these, 1 wholly recovered and the other 2 partly. Muscular rigidity is noted in 5 cases, of which 2 wholly recovered, but it was undoubtedly present in many others. The latter symptoms mean much damage to the cord, and the wonder is that any recover from them. *Where the paralysis came on while the patient was under treatment* (19 *cases*), *the percentage of recoveries was one hundred in the* 17 *cases whose termination was known.* Of the 2 cases where the termination was doubtful, 1 was recovering power quite fast at the end of six months, and the other was still paralyzed when two years had gone by. Neither has been heard from since.

The treatment in all cases was directed to the spinal caries, and was protective, viz.: the application of the Taylor spinal apparatus, and chin-piece when necessary. Great stress was laid on thorough and adequate mechanical support entirely under the surgeon's control, and modified by him to suit the varying indications. The protection was only considered satisfactory when, with a firm and even impression of the pad-plates, the patient's

acute symptoms subsided in a few days or weeks, his health and strength improved, and the progress of the deformity was substantially arrested without discomfort from the apparatus. The patient was put to bed and not encouraged to walk until there was a pretty complete return of power. There was no medicinal treatment directed to the paralysis.

It seems justifiable to assume from the statistics that in the course of a few months the paralysis will disappear without any other than the protective treatment for Pott's disease joined to rest in bed during the time of disability. Moreover, when paralysis occurs while the spine is being efficiently supported, our study shows that its duration is shortened, its type is milder, complications are fewer, and complete recovery from it is almost certain. Lastly, its rare occurrence during adequate mechanical treatment is to be noted. In these statistics it occurred only 19 times in 445 cases. Gibney[1] reported 62 cases in 295 cases of Pott's disease at the Hospital for the Ruptured and Crippled, of which perhaps a third were paralyzed before coming to the hospital. The French treatises deal with the disease as seen in hospital wrecks, and afford us no information as to its course or the frequency of its occurrence.

It seems, then, to be an affection of rare occurrence in Pott's disease under efficient protective treatment. It occurs without regard to the amount or character of the deformity, and is usually preceded by much pain; on the average it lasts a little less than a year. Its prognosis is extremely favorable in mild cases, or in severe ones if they can be treated early. If the bladder and rectum are involved, or muscular rigidity of long standing is present, the prognosis is not so good; but even then it is far from being bad. The treatment is efficient mechanical support to the spine and rest in bed. Under

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[1] Journal of Nervous and Mental Diseases, April, 1878.

these circumstances it becomes a complication of spinal caries of no very serious import.

NOTE.—In order to make this paper as complete as possible we quote the following from a letter from Dr. C. Fayette Taylor, in Meran, Austria :

" You have chosen a good subject for your article, but it is probable that I could supply from memory the final results in some cases which failed to be recorded in the books. Cases that came paralyzed—long-standing cases, I mean—and cases that discontinued treatment after insufficient time and opportunity, ought not to be considered as having any bearing on the general results of paralysis under treatment by protection ; for by protection is necessarily meant adequate protection, as to efficiency, time of beginning, continuing, and terminating it. Considered in this light, I am not aware that there was ever a case under my treatment, to whom protection was given at any time before the cessation of the destructive process, whether paralyzed at the time it came under observation or after, which did not entirely and permanently recover. In that connection some strange things have happened, leading me to believe that paralysis occurring during the course of disintegration of vertebræ is always in the first instance in consequence of the pressure of fluid, but that fluid-pressure does not produce permanent injury to the cord, merely suspending its function—that is to say, sufficient fluid-pressure to produce disintegration of the cord is not reached because the surrounding soft tissues give way first and the fluid escapes into the adjacent parts, thus diminishing the pressure on the cord. Hence the frequent occurrence of the appearance of a lumbar or psoas abscess and the recovery from the paralysis at the same time. More commonly, however, the retarded disintegration and diminished formation of fluid consequent on protecting the carious vertebræ allows the fluid to be absorbed and so lessens the pressure, when the cord resumes its function and the paralysis ceases. Permanent paralysis in consequence of Pott's

disease, the evidence of my experience goes to show, is nearly always the result of bone-pressure from narrowing of the spinal canal—not often from exostoses, as is frequently claimed. The kyphosis results of course from destruction of several vertebral bodies. Even in the latter case, when the cord is bent at a sharp angle in a narrowed canal, there is good reason to believe that in some the cavity enlarges in time so as to give the cord more room, and its function becomes wholly or partly restored. This probability is forcibly illustrated in several cases of traumatism, where the paralysis occurred immediately.

"D—— fell out of a third-story window and was taken up totally paralyzed in his lower extremities. I saw him nine months later. At this time his lumbar spine presented every appearance of an ordinary case of Pott's disease, several vertebræ being affected and the lumbar portion bulging outward in the ordinary way after loss of substance in the bodies. But there had lately been a gain of some motion in the lower extremities, which I attributed to enlargement of the spinal canal consequent on the softening of the bodies. He was walking about in a month and recovered rapidly and entirely under protection. The peasant here—mentioned in a previous letter—is another apparently precisely similar example, and seems likely to have a similar result. He was run over last July, and taken up paralyzed in his lower extremities. I saw him a month ago, and already there was slight power in one leg, though disintegration was evidently going forward in the bodies of the vertebræ, as indicated by the rational symptoms and the appearance of the projection in the back. If, as seems probable, there was dislocation or fracture by the injury in these cases, causing pressure on the spinal cord, we can only account for even the slightest improvement in its function by supposing a diminution of the pressure."